

**An Investigation of the Overlap among Disinhibited Eating Behaviors
in Children and Adolescents**

by

Anna Vannucci

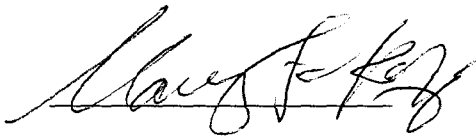
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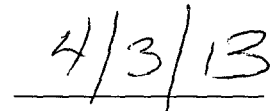
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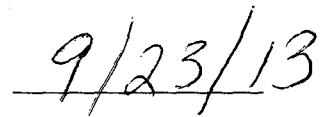


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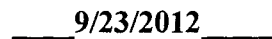


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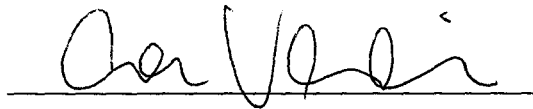
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Title of Thesis: An Investigation of the Overlap among Disinhibited Eating Behaviors in Children and Adolescents

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Eating- and weight-related problems represent a significant public health threat among children and adolescents. The identification of common risk factors promoting obesity and eating disorders in youth may enhance prevention and early intervention efforts. Three pediatric disinhibited eating behaviors—eating in the absence of hunger, emotional eating, and loss of control eating—appear to elevate risk for eating disorder psychopathology, general psychopathology, and weight problems among children and adolescents. Despite their theoretical similarities, studies have yet to examine the overlap among all three disinhibited eating behaviors between youth. A systematic evaluation may lead to the identification of behavioral risk profiles that providers may be able to screen for and make predictions about based on correlates of these subtypes. Therefore, the objectives of the current study were to: 1) determine whether children and adolescents can be classified into subtypes based on the overlap of disinhibited eating behaviors; and 2) investigate the validity and clinical utility of subtypes based on the overlap of disinhibited eating behaviors among children and adolescents.

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Chapter 1: Introduction

Problem of Eating and Weight Disorders

Eating- and weight-related problems represent a significant public health threat among children and adolescents. Recent prevalence estimates indicate that over one-third of children in the United States are overweight (body mass index, kg/m^2 , BMI, $\geq 85^{\text{th}}$ percentile) or obese ($\geq 95^{\text{th}}$ BMI percentile) (Ogden, Carroll, Kit, & Flegal, 2012). There are profound racial/ethnic differences in the population prevalence of pediatric obesity; recent estimates indicate that approximately 39% of Hispanic/Latino and non-Hispanic black youth between the ages of 2 and 19 years are overweight or obese as compared to 28% of non-Hispanic white youth (Ogden, et al., 2012). Not only are there more obese children now than in the past, but the severity of overweight (rates of extreme pediatric obesity, $\text{BMI} \geq 99^{\text{th}}$ percentile) among these children is greater (Freedman, Kettel Khan, Serdula, Ogden, & Dietz, 2006; Koebrick et al., 2010). Over the past thirty years, there have been disproportionate increases in the prevalence of obesity, especially extreme pediatric obesity, among non-Hispanic black and Hispanic/Latino children as compared to non-Hispanic white children (Freedman, et al., 2006; Koebrick, et al., 2010).

Disordered eating in children and adolescents appears to be on the rise as well. While the lifetime prevalence of eating disorders (i.e., anorexia nervosa, bulimia nervosa, and binge eating disorder) among youth is estimated to be 3%, and subclinical eating disorders (i.e., subthreshold binge eating disorder and bulimia nervosa, eating disorder not otherwise specified) are reported by approximately 3% of children and adolescents (Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011), subthreshold disordered eating (i.e., binge eating, unhealthy weight control behaviors such as excessively restrictive dieting or driven exercise) rates may be much higher.

Despite the assumption that eating disorders occur primarily among Caucasians, eating disorders and subthreshold disordered eating patterns are reported in equal rates in non-Hispanic white, non-Hispanic black, and Hispanic/Latino youth (Swanson, et al., 2011). The prevalence of pediatric disinhibited eating behaviors linked to eating disorders and obesity—such as loss of control eating, emotional eating, and eating in the absence of hunger—may be much higher as they have been reported in 3 to 60% of children and adolescents (Moens & Braet, 2007; Shapiro et al., 2007; Tanofsky-Kraff, 2008; Tanofsky-Kraff, Theim, et al., 2007). High rates of disinhibited eating behaviors have been found among youth reporting disordered eating behaviors that have also been shown to promote obesity, such as binge eating and dieting.

Pediatric obesity and disordered eating, at clinical and subclinical levels, are associated with significant psychiatric and medical comorbidity and a markedly reduced quality of life (Barzin, Hosseinpanah, Fekri, & Azizi, 2011; Madden, Morris, Zurynski, Kohn, & Elliot, 2009; Must & Strauss, 1999; Swanson, et al., 2011; Wardle & Cooke, 2005; Weiss et al., 2004). Moreover, obese youth and those diagnosed with eating disorders report greater healthcare utilization (Hampl, Carroll, Simon, & Sharma, 2007; Mitchell et al., 2009; Striegel-Moore et al., 2008; Wang & Dietz, 2002), which is consistent with the well-documented economic burden related to obesity and eating disorders (Finkelstein, Trogdon, Cohen, & Dietz, 2009; Robergeau, Joseph, & Silber, 2006; Simon, Schmidt, & Pilling, 2005; Trassande & Chatterjee, 2009; Trassande, Liu, Fryer, & Weitzman, 2009). These findings highlight the serious problem of eating and weight disorders in children and adolescents, and underscore the need to illuminate the characteristics that promote excessive weight gain and eating pathology.

Although the etiologies of eating disorders and obesity have often been examined independently, there is an increasing recognition that the identification of common risk factors

promoting obesity and eating disorders in youth may enhance prevention and early intervention efforts (Austin, 2011; Neumark-Sztainer, 2005). Research has identified several risk factors common to both eating disorders and obesity, including high restraint or unhealthy dieting practices, exposure to the media, body dissatisfaction, and the experience of weight-related teasing (Haines & Neumark-Sztainer, 2006). Despite the likely centrality of aberrant eating behavior to development of obesity and eating disorders, there has been little focus on the identification of common modifiable behaviors that confer increased vulnerability for eating- and weight-related problems. Elucidating such behavioral risk factors is crucial to the design and implementation of successful, targeted prevention and early intervention strategies.

Theoretical Frameworks of Disinhibited Eating

Disinhibited eating—bouts of overeating prompted by feelings of letting go or a lack of self-regulation (Heatherton & Baumeister, 1991; Herman & Polivy, 1980)—may represent an important set of modifiable behaviors that elevate risk for eating and weight-related problems among children and adolescents (Shomaker, Tanofsky-Kraff, & Yanovski, 2010). Disinhibited eating is related to a diminished or complete inability for individuals to initiate eating only when they are hungry and terminate eating when they are satiated (Bryant, King, & Blundell, 2007).

Several theoretical approaches have proposed distinct mechanisms leading to the onset and maintenance of disinhibited eating behaviors. Schachter's (1968) externality theory of obesity posits that bouts of overeating result from a poor responsiveness to physiological, internal cues for hunger and fullness and high responsiveness to environmental, external cues such as smell and taste. Several lines of interrelated, contemporary research have built upon Schachter's (1968) externality theory of obesity and reconceptualized the "obese eating style" as

a lack of satiety responsiveness, defined as the extent to which an individual's eating behavior tends to correspond to internal, physiological satiety signals (Wardle, Guthrie, Sanderson, & Rapoport, 2001). Psychosomatic theory proposes that individuals who regularly engage in disinhibited eating behaviors have interoceptive awareness deficits, which make it difficult to distinguish somatic sensations related to the experience of emotions from those associated with appetite (Bruch, 1973). Further expanding upon Bruch's (1973) psychosomatic theory, recent affective theories suggest that disinhibited eating occurs during times when food is sought out to alleviate, escape, or provide comfort from negative affective states (Dubé, LeBel, & Lu, 2005; Heatherton & Baumeister, 1991; Macht & Simons, 2000).

Disinhibited Eating Behaviors: Associations with Body Weight and Eating Pathology

Research has revealed the presence of several observable types of disinhibited eating behaviors among youth, including eating in the absence of hunger, emotional eating, and loss of control eating (Shomaker, Tanofsky-Kraff, & Yanovski, 2010). Theoretically, eating in the absence of hunger most closely aligns with Schachter's externality theory of obesity and the notion of a lack of satiety responsiveness. Emotional eating and loss of control eating, on the other hand, has typically been conceptualized using the framework of psychosomatic and affective theories. Eating in the absence of hunger, emotional eating, and loss of control eating are conceptually distinct and have been distinguished from each other in laboratory paradigms and when examining retrospective reports of these behaviors. This section defines each disinhibited eating behavior and reviews literature on its association with body weight and eating pathology.

Eating in the Absence of Hunger.

Eating in the absence of hunger refers to eating in response to the presence of palatable foods in the absence of physiological hunger (Kral & Faith, 2007), and estimates suggest that approximately 60% of youth exhibit eating in the absence of hunger to at least some degree (Moens & Braet, 2007). Eating in the absence of hunger is often measured directly by observing children's *ad libitum* energy intake following the consumption of a meal or snack to satiety (Birch, Fisher, & Davison, 2003; Fisher & Birch, 2002; Shomaker, Tanofsky-Kraff, Zocca, et al., 2010), but can also be assessed by children's and parent's self-reports (Tanofsky-Kraff et al., 2008; Zocca et al., 2011). Satiety responsiveness—the construct that theoretically underlies eating in the absence of hunger—has also been assessed by parent self-report questionnaires (Parkinson, Drewett, Le Couteur, & Adamson, 2010; Wardle, et al., 2001).

Cross-sectional studies of eating in the absence of hunger among children and adolescents have demonstrated a positive association with overweight status in controlled laboratory paradigms (Fisher & Birch, 2002; Fisher et al., 2007; Shomaker, Tanofsky-Kraff, Zocca, et al., 2010) and in naturalistic settings (Cutting, Fisher, Grimm-Thomas, & Birch, 1999; Hill et al., 2008; Moens & Braet, 2007). Among children between 8 and 18 years old, eating in the absence of hunger was self-reported more frequently among obese as compared to non-obese youth (Tanofsky-Kraff, et al., 2008). Parental reports of satiety responsiveness have also been shown to be associated with BMI standard scores among children between 3 and 13 years (Carnell & Wardle, 2008; Sleddens, Kremers, & Thijs, 2008; Viana, Sinde, & Saxton, 2008). Longitudinal studies indicate that the degree of eating in the absence of hunger (overall energy intake after consuming a meal to satiety) may increase with age (Birch, et al., 2003; Fisher, et al., 2007), but there have been few prospective studies reporting on the relationship between eating

in the absence of hunger and excess weight gain. In a sample of Hispanic children and adolescents, eating in the absence of hunger predicted weight gain a year later after adjusting for age, sex, and puberty, but the effect was attenuated after accounting for baseline BMI status (Butte et al., 2007).

There has been a paucity of research examining the relationship between eating in the absence of hunger and eating pathology and psychological problems in youth. However, one longitudinal study examined behavioral predictors of eating in the absence of hunger. Birch and colleagues found that maternal restriction of their girls' food intake at age 7 predicted increases in energy consumed in the absence of hunger at age 9 (Birch, et al., 2003). This relationship was especially robust among overweight girls exposed to maternal feeding restriction. Some early studies suggest that young women with anorexia nervosa and bulimia nervosa report more impaired satiety responsiveness than healthy controls (Heilbrun & Worobow, 1990, 1991). Additionally, one study found a positive association between self-reported eating in the absence of hunger and symptoms of anxiety and depression in non-treatment seeking children and adolescents (Tanofsky-Kraff, et al., 2008). Overall, these preliminary findings suggest that it may be worthwhile to further examine the relationship between eating in the absence of hunger and eating pathology in youth.

Emotional Eating.

Emotional eating refers to consuming food in an attempt to cope with transient or enduring negative emotions (Faith, Allison, & Geliebter, 1997). While emotional eating appears to be relatively uncommon among young children (Carper, Fisher, & Birch, 2000; van Strien & Bazelier, 2007; van Strien & Oosterveld, 2008; Wardle, et al., 2001), over 50% of youth in middle childhood and adolescence at least occasionally report emotional eating (Shapiro, et al.,

2007; Tanofsky-Kraff, Theim, et al., 2007). Emotional eating primarily has been assessed by children's or parent's self-reports (Braet & van Strien, 1997; Rollins et al., 2011; Tanofsky-Kraff, Theim, et al., 2007; Wardle, et al., 2001). Although less common, pediatric emotional eating has been assessed in laboratory settings by measuring energy intake following a negative mood induction (Goldschmidt, Tanofsky-Kraff, & Wilfley, 2011) or reports of pre-meal state negative affect (Vannucci et al., 2011).

Several cross-sectional studies have shown that parental and child reports of emotional overeating are associated with overweight and obese status (Braet et al., 2008; Braet & van Strien, 1997; Parkinson, et al., 2010; Sleddens, et al., 2008; Webber, Hill, Saxton, Van Jaarsveld, & Wardle, 2009). Other studies examining youths' emotional eating and body weight have yielded null effects (Caccialanza et al., 2004; Tanofsky-Kraff, Theim, et al., 2007; van Strien & Bazelier, 2007). Although there have been no prospective studies of pediatric emotional eating, self-reported emotional eating in adult twins was associated with retrospective reports of weight gain from early to middle adulthood (Sung, Lee, & Song, 2009). While the relationship between emotional eating and body weight is not definitive, some (Braet & van Strien, 1997; Nguyen-Rodriguez, Chou, Unger, & Spruijt-Metz, 2008; Rollins, et al., 2011; Vannucci, et al., 2011; Wardle et al., 1992), though not all (van Strien & Oosterveld, 2008), findings suggest that emotional eating may be linked with aberrant eating patterns—excess overall energy intake and consumption of high-fat foods—that could contribute to excessive weight gain over time.

Emotional eating has also been associated with elevated eating pathology and psychological dysfunction in youth. Research suggests that there is a positive association between emotional eating and body dissatisfaction and a drive for thinness, as well as concerns about eating, weight, and shape in adolescents (Goldschmidt, Aspen, Sinton, Tanofsky-Kraff, &

Wilfley, 2008). In a study comparing a group of adolescents with eating disorders and healthy adolescents from the community, the eating disorder group reported a greater frequency of emotional eating than the healthy controls (van Strien, Engels, Leeuwe, & Snoek, 2005). In addition, several studies demonstrated that emotional eating was positive correlated with depressive and anxiety symptoms, perceived stress, and internalizing and externalizing behavior problems among children and adolescents (Braet & van Strien, 1997; Nguyen-Rodriguez, et al., 2008; Tanofsky-Kraff, Theim, et al., 2007; van Strien, et al., 2005).

Loss of Control Eating.

Binge eating is defined as overeating an objectively large amount of food while experiencing a lack of control over what or how much is being eaten (American Psychiatric Association [APA], 2000). Loss of control eating is characterized by the subjective experience of being unable to control what or how much one is eating, regardless of the amount of food consumed (Tanofsky-Kraff, Goossens, et al., 2007; Tanofsky-Kraff et al., 2004). Loss of control eating, by definition, encompasses binge eating episodes and may be a preferable construct for use in pediatric samples due to the difficulties of assessing binge eating in youth (Goldschmidt, Aspen, et al., 2008; Wilfley, Vannucci, & White, 2010) and recent data suggesting that there is no meaningful difference between objectively and subjectively large binge episodes in children (Shomaker, Tanofsky-Kraff, Elliott, et al., 2010).

The prevalence of loss of control eating ranges from 4% to 45%, with higher estimates among overweight youth (versus non-overweight), adolescents (versus pre-adolescents), and when assessed via questionnaire (versus semi-structured interview) (Tanofsky-Kraff, 2008). Several interview-based (Bryant-Waugh, Cooper, Taylor, & Lask, 1996; Tanofsky-Kraff, Goossens, et al., 2007), self-report (Goldschmidt, Doyle, & Wilfley, 2007), and laboratory

methods (Hilbert, Tuschen-Caffer, & Czaja, 2010; Tanofsky-Kraff, McDuffie, et al., 2009) have been used to assess loss of control eating. These reports have consistently shown that children with reported loss of control eating differ from those without loss of control eating on a number of physical, behavioral, and psychological indices.

Findings have demonstrated a relationship between pediatric loss of control eating and body weight. In cross-sectional studies of children and adolescents, those who reported loss of control eating were more likely to be overweight and have greater body fat mass than youth who reported no loss of control eating episodes (Ackard, Neumark-Sztainer, Story, & Perry, 2003; Field et al., 1999; Field, Colditz, & Peterson, 1997; Neumark-Sztainer et al., 1997; Shomaker, Tanofsky-Kraff, Elliott, et al., 2010; Tanofsky-Kraff, Goossens, et al., 2007; Tanofsky-Kraff, et al., 2004). Observational studies of children and adolescent's eating in the laboratory suggest that youth reporting loss of control eating consumed more overall energy (Hilbert & Czaja, 2009; Hilbert, et al., 2010), especially from fat and carbohydrate (Hilbert, et al., 2010; Tanofsky-Kraff, McDuffie, et al., 2009)—behavioral patterns that might promote excessive weight gain. These laboratory findings appear to generalize to the natural environment (Hilbert, Rief, Tuschen-Caffier, de Zwaan, & Czaja, 2009). Prospective studies have found that the presence of loss of control eating predicts excessive weight gain (Tanofsky-Kraff, Yanovski, et al., 2009), greater gains in body fat mass (Tanofsky-Kraff et al., 2006), and the onset of obesity (Field et al., 2003; Stice, Cameron, Killen, Hayward, & Taylor, 1999; Stice, Presnell, & Spangler, 2002).

Cross-sectional studies of children and adolescents with loss of control eating have consistently reported elevated eating disorder psychopathology—including cognitive restraint and concerns about eating, weight, and shape—than youth without loss of control eating (Goossens, Braet, & Decaluwe, 2007; Hartmann, Czaja, Rief, & Hilbert, 2010; Tanofsky-Kraff,

Faden, Yanovski, Wilfley, & Yanovski, 2005; Tanofsky-Kraff, Goossens, et al., 2007). Negative affect—marked by symptoms of depression and anxiety and behavioral disturbance—has also been consistently associated with loss of control eating in children and adolescents (Goldschmidt et al., 2008; Shomaker, Tanofsky-Kraff, Elliott, et al., 2010; Stice, et al., 2002; Tanofsky-Kraff, et al., 2005). Loss of control eating also prospectively predicted the emergence of full- and partial-syndrome binge eating disorder and the worsening of disordered eating attitudes (Tanofsky-Kraff et al., 2011). Additionally, baseline reports of lifetime loss of control among youth predicted increases in anxiety, while persistent loss of control eating over time predicted worsening depressive symptoms in children and adolescents (Tanofsky-Kraff, et al., 2011).

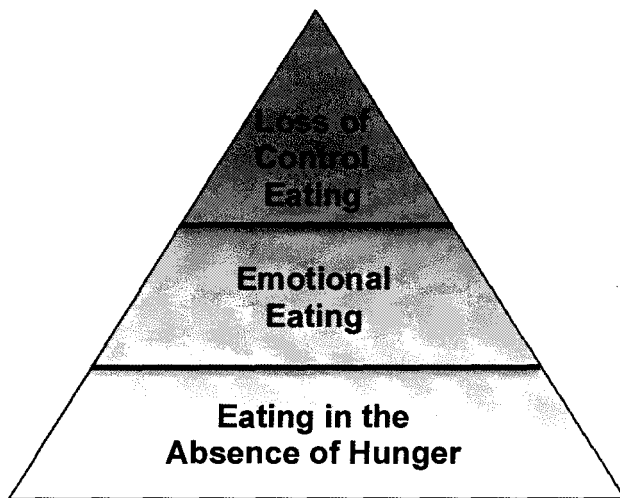
Overlap Among Disinhibited Eating Behaviors

Disinhibited eating behaviors are characterized by a lack of healthy regulation over food intake, and include eating in the absence of hunger, emotional eating, and loss of control eating (Shomaker, Tanofsky-Kraff, & Yanovski, 2010). Overall, findings suggest that eating in the absence of hunger, emotional eating, and loss of control eating may be associated with overweight status and disordered eating pathology in children and adolescents. Therefore, it is possible that these disinhibited eating behaviors may represent a range of modifiable behaviors that elevate risk for eating and weight-related problems. While eating in the absence of hunger, emotional eating, and loss of control eating are distinct constructs (as defined conceptually and in laboratory studies described above), they are similar in that youth with disinhibited eating behaviors are unable to initiate eating only when they are hungry and/or terminate eating when they are full (Shomaker, Tanofsky-Kraff, & Yanovski, 2010). Despite these commonalities,

eating in the absence of hunger, emotional eating, and loss of control eating primarily have been examined as separate entities within studies.

It has been proposed that disinhibited eating behaviors are likely organized in a pyramidal fashion (**Figure 1**) (Shomaker, Tanofsky-Kraff, & Yanovski, 2010), which will be referred to here as the pyramid model of disinhibited eating. This pyramid model proposed that disinhibited eating behaviors are ordered from bottom to top based upon prevalence, pathology, and overlap. In contrast, emotional eating and eating in the absence of hunger do not necessarily involve feelings of loss of control over eating. Eating in the absence of hunger—at the bottom of the pyramid—is hypothesized to be the most prevalent and least pathological disinhibited eating behavior reported among children and adolescents. Emotional eating is thought to be slightly less common and somewhat more pathological, while loss of control eating—at the top of the pyramid—is proposed to occur among the smallest subset of youth and to be associated with the most severe psychological and weight-related associations and outcomes. Also important to this conceptualization is that behaviors at the top of the pyramid (loss of control eating) often overlap with and involve behaviors in the bottom half of the pyramid (emotional eating or eating in the absence of hunger). For example, a teenager with loss of control eating very likely engages in eating in response to negative affective triggers, and she or he also is likely to eat when not physiologically hungry. In contrast, behaviors at the bottom (eating in the absence of hunger) do not necessarily involve behaviors located above them (emotional eating, loss of control eating). Given that disinhibited eating behaviors have been theoretically and empirically distinguished from each other, it is important to note that the pyramid model *does not* suggest a dimensional framework whereby eating in the absence of hunger, emotional eating, and loss of control eating

Figure 1. Pyramid Model of Disinhibited Eating.



Note: The Pyramid Model of Disinhibited Eating (Shomaker, Tanofsky-Kraff, & Yanovski, 2010) suggests that disinhibited eating behaviors are ordered from bottom to top based upon prevalence, pathology, and overlap. Eating in the absence of hunger—at the bottom of the pyramid—is hypothesized to be the most prevalent and least pathological disinhibited eating behavior reported among children and adolescents. Emotional eating is thought to be slightly less common and somewhat more pathological, while loss of control eating (both objective and subjective binge episodes) —at the top of the pyramid—is proposed to occur among the smallest subset of youth and to be associated with the most severe psychological and weight-related associations and outcomes. Also important to the pyramid model, is that behaviors at the top (loss of control eating) often include behaviors in lower parts of the pyramid (emotional eating and eating in the absence of hunger). In contrast, behaviors at the bottom (eating in the absence of hunger) do not necessarily involve behaviors located above them (e.g., emotional eating and loss of control eating).

lie on a continuum. Therefore, the pyramid model provides a framework for understanding the clinical implications of individual differences in the overlap of these behaviors in the population.

Consistent with the pyramid model of disinhibited eating behaviors, emerging data point to the significant overlap among measures of eating in the absence of hunger, emotional eating, and loss of control eating in youth. Self-reported emotional eating in pediatric samples was associated with a higher frequency of self-reported eating in the absence of hunger (Tanofsky-Kraff, et al., 2008) and increased energy intake in the absence of hunger in the laboratory (Moens & Braet, 2007). Emotional eating has also been found to prospectively predict binge eating onset in adolescent girls (Stice, et al., 2002). There is also some evidence in cross-sectional studies to suggest that children and adolescents with loss of control eating report a greater frequency of eating in the absence of hunger and emotional eating than those without loss of control eating (Goossens, et al., 2007; Shapiro, et al., 2007; Shomaker, Tanofsky-Kraff, Elliott, et al., 2010; Tanofsky-Kraff, Goossens, et al., 2007; Tanofsky-Kraff, et al., 2008; Tanofsky-Kraff, Theim, et al., 2007). Yet, despite their overlap, eating in the absence of hunger also can be clearly differentiated from loss of control eating when examining associations between child and parent self-reports of these behaviors (Zocca, et al., 2011). While these findings are promising, studies have yet to systematically examine individual differences the overlap among all three disinhibited eating behaviors between youth. Further exploration of individual differences in the overlap among all three disinhibited eating behaviors is warranted.

As previously stated, the pyramid model of disinhibited eating hypothesizes that the combination of having more than one type of disinhibited eating behavior is likely to be related to greater risk for psychological problems, eating pathology, and potentially excessive weight gain (Shomaker, Tanofsky-Kraff, & Yanovski, 2010). For example, overweight children with

loss of control eating consumed more fat in response to a negative affect induction—a laboratory construct of emotional eating—as compared to those without loss of control eating (Goldschmidt, et al., 2011). Although self-reports of emotional eating or eating in the absence of hunger were not assessed, these data provide some preliminary support for the notion that overlap of disinhibited eating behaviors are associated with more aberrant eating behaviors. Yet, whether such co-morbidity of disinhibited eating behaviors is tied to risk for excess weight gain or greater eating disorder and general psychopathology in youth remains to be determined.

Rationale for the Current Study

The increasing prevalence of pediatric eating disorders and obesity constitute a significant public health problem in the United States. Given the cost and challenges of treating full syndrome eating disorders and the high recidivism associated with childhood obesity treatments, a focus on prevention and targeted early intervention efforts is warranted. Despite the commonalities in the etiology of disordered eating and obesity (i.e., unhealthy regulation of eating and activity), most research to date has examined risk factors and prevention targets for eating disorders and obesity independently of one another. There is an increasing recognition that eating disorders and obesity prevention efforts may be the most successful if they capitalize on the commonalities underlying eating- and weight-related problems (Austin, 2011; Neumark-Sztainer, 2005). Moreover, a focus on shared risk factors that promote both eating disorders and obesity may be cost-effective (Austin, 2011; Neumark-Sztainer, 2005).

Reports from National Institutes of Health Workshops on the development of research priorities in eating disorders and obesity noted that greater attention to phenotype characterization is needed to improve prevention efforts and intervention development (Grilo,

Cachelin, & Yanovski, 1997; NHLBI, 2007). Therefore, research is needed to examine individual differences in three disinhibited eating behaviors shown to be associated with adverse eating and weight related outcomes and correlates—eating in the absence of hunger, emotional eating, and loss of control eating. An accurate description and conceptualization of disinhibited eating behaviors and how they overlap is necessary to permit consideration of distinct behavioral risk profiles that contribute to shared or unique eating disorder and obesity risk in youth. An improved characterization of individual differences in the overlap and correlates of disinhibited eating behaviors would have important clinical implications: 1) (potentially) evaluate support for the pyramid model of disinhibited eating that could form the basis for the development of novel interventions; and 2) enhance the ability of clinicians to make meaningful predictions based on the overlap of disinhibited eating behaviors in youth.

Chapter 2: Specific Aims and Hypotheses

Disinhibited eating behaviors—including eating in the absence of hunger, emotional eating, and loss of control eating—appear to be an important set of modifiable behaviors that elevate risk for eating and weight-related problems in youth. The identification of distinct behavioral subtypes that confer greater vulnerability for developing eating disorders and obesity among children and adolescents would assist with targeting at-risk groups for prevention programs and improve the development of tailored interventions. Preliminary work supports the utility of subtyping youth with regard to obesity-related risk behaviors (Boone-Heinonen, Gordon-Larsen, & Adair, 2008; Huh et al., 2011). Recent evidence also suggests that it is clinically meaningful to subtype obese adolescents based on the presence of loss of control eating (Sysko, Zakarin, Devlin, Bush, & Walsh, 2011)—a key disinhibited eating behavior. However, no prior studies have attempted to use multiple behaviors associated with both eating disorders and body weight to examine at-risk subtypes among children and adolescents. Therefore, the objective of the current study is to investigate the range of individual differences based on the overlap of three disinhibited eating behaviors in children and adolescents.

Specific Aim 1: To characterize subtypes based on the overlap of disinhibited eating behaviors in children and adolescents.

Hypothesis 1.1: Based on the pyramidal model of disinhibited eating behaviors, it is hypothesized that three distinct subtypes based on disinhibited eating behaviors will be identified, including: 1) an asymptomatic group (low on all three disinhibited eating behaviors), 2) a low disinhibited eating group characterized by high eating in the absence of hunger and emotional eating but low loss of control eating, 3) and a high disinhibited eating group characterized by being high on all three disinhibited eating behaviors.

Specific Aim 2: To investigate the validity and clinical utility of subtypes based on the overlap of disinhibited eating behaviors among children and adolescents.

Hypothesis 2.1: Based on the pyramidal model of disinhibited eating behaviors, it is hypothesized that the behavioral subtypes based on disinhibited eating will be distinguished by their adiposity, such that the high disinhibited eating group will have the highest BMI standard scores and percent fat mass followed by the low disinhibited eating group and asymptomatic group, respectively.

Hypothesis 2.2: Based on the pyramidal model of disinhibited eating behaviors, it is hypothesized that the high disinhibited eating group will report greater eating pathology (restraint, concerns about eating, shape, and weight) and general psychopathology (depressive and anxiety symptoms) than the low disinhibited eating group, and both disinhibited eating groups will report greater pathology than the asymptomatic group.

Hypothesis 2.3: Based on the pyramidal model of disinhibited eating behaviors, it is hypothesized that behavioral subtypes based on disinhibited eating will be distinguished based on eating behavior in the laboratory, such that the low and high disinhibited eating groups will consume more overall energy intake and intake from fat and carbohydrate at a test meal designed to capture disinhibited eating as compared to the asymptomatic group.

Chapter 3: Methods

Participants

Participants were children and adolescents (8-18 years) who were enrolled in non-intervention protocols, and adolescent girls (12-17 years) participating in a randomized controlled trial examining the efficacy of an excess weight gain prevention program. All participants were recruited through flyers posted on public bulletin boards at the National Institutes of Health (NIH), local libraries, supermarkets, and school parent email listservs in the Washington, DC greater metropolitan area. For the prevention study, additional recruitment efforts involved advertisements in local newspapers as well as mailings to parents in Montgomery County, Prince George's County, and local Maryland school districts.

For the non-intervention studies, boys and girls who had a BMI greater than or equal to the 5th percentile for age and sex were eligible for participation. Individuals were excluded if they had a significant medical condition; had abnormal hepatic, renal, or thyroid function; were taking medications known to affect body weight; experienced a weight loss of more than five pounds (2.3 kg) in the past three months; were undergoing weight loss treatment; or had a psychiatric disorder that might impede protocol compliance. For the prevention study, girls were studied prior to participation in the interventions. By study design, participants were female, 12 to 17 years, had a BMI between the 75th to 97th percentile, and reported the presence of loss of control eating in the previous month. Girls were excluded if they had a significant medical condition; an obesity-related medical complication that would require a more aggressive weight loss intervention; a current psychiatric disorder that might impede protocol compliance; history of an eating disorder (other than binge eating disorder); a loss of greater than 3% of body weight

in the previous 3 months; were taking medication known to impact body weight or psychiatric medications; or undergoing weight loss treatment or currently participating in psychotherapy.

Procedures

For the non-intervention studies, potential participants and a parent/guardian were seen at the NIH Hatfield Clinical Research Center. For the prevention study, interested families were screened by telephone and participated in two screening assessments at USUHS and NIH on separate days. For both studies, participants completed self-report questionnaires and a semi-structured interview regarding eating disorder psychopathology. Additionally, fasting anthropometric measures were taken. A subset of participants ($n = 223$) from the non-intervention study protocol was scheduled for laboratory test meal visits as described previously (Tanofsky-Kraff, McDuffie, et al., 2009). All visits took place following an overnight fast. Informed parental consent and child assent were obtained for both studies.

Measures

Eating in the Absence of Hunger. The Parent Report version of the Eating in the Absence of Hunger Questionnaire, which is a parallel version to the Eating in the Absence of Hunger Questionnaire for Children (Tanofsky-Kraff, et al., 2008), is a 14-item parent-report questionnaire designed for use with 8-18 year olds that assesses the frequency with which youth eat when they are either not hungry or already sated. Respondents rate the frequency that they eat in the absence of hunger on a 5-point scale ranging from “Never” through “Always”. The Parent Report version of the Eating in the Absence of Hunger Questionnaire generates three subscales that reflect distinct aspects of eating in the absence of hunger, including: 1) External eating; 2)

Negative affect; and 3) Fatigue/boredom; it also generates a total score created from averaging the three subscale scores. This questionnaire has demonstrated good internal consistency, temporal stability, and convergent validity with loss of control eating, and temporal stability for all scales (Zocca, et al., 2011). The Parent Report version, as opposed to the child report version was used, since only parent-reports appear to demonstrate construct validity with laboratory meals designed to capture eating in the absence of hunger in response to external cues in non-intervention seeking youth similar to the current sample (Shomaker et al., under submission).

Emotional Eating. The Emotional Eating Scale Adapted for Children and Adolescents, designed for use with 8-18-year-olds (Tanofsky-Kraff, Theim, et al., 2007) was adapted from the Emotional Eating Scale for adults (Arnow, Kenardy, & Agras, 1995). The Emotional Eating Scale Adaptive for Children and Adolescents is a 25-item self-report measure used to assess the propensity to cope with negative affect by eating. Respondents rate their desire to eat in response to each emotion on a 5-point scale from “I have no desire to eat” through “I have a very strong desire to eat.” The Emotional Eating Scale Adapted for Children and Adolescents generates three subscales reflecting the urge to eat in response to: 1) Anger, anxiety, and frustration, 2) Depressive symptoms, and 3) Feeling unsettled. In addition, summing of the individual items generates a total score (Vannucci, et al., 2011). The Emotional Eating Scale Adapted for Children and Adolescents has demonstrated good psychometric properties (Tanofsky-Kraff, Theim, et al., 2007) and construct validity (Vannucci, et al., 2011).

Loss of Control Eating and Eating Disorder Psychopathology. The Eating Disorder Examination version 12.0D/C.2 (EDE; Fairburn & Cooper, 1993) or the child version (Bryant-Waugh, Cooper, Taylor, & Lask, 1996) was used to determine the presence or absence of objective binge episodes (OBE; consumption of an unambiguously large amount of food with a

sense of loss of control) and subjective binge episodes (SBE; the experience of loss of control while consuming an ambiguously large amount of food). Consistent with prior research on loss of control eating in pediatric samples (Glasofer et al., 2007; Shomaker, Tanofsky-Kraff, Elliott, et al., 2010; Tanofsky-Kraff, Goossens, et al., 2007; Tanofsky-Kraff, McDuffie, et al., 2009; Tanofsky-Kraff, et al., 2004), the presence or absence of OBEs and SBEs in the past month—rather than the past three months—was determined. The presence or absence of loss of control eating in the past month was coded if youth reported any OBEs, any SBEs, or both. Additionally, this threshold was determined for the current study because it is thought that children can more reliably recall episodes that occurred recently versus several months prior and the proximal timeframe was more consistent with the self-report questionnaires of disinhibited eating.

The EDE has had good interrater reliability for all episode types (Spearman correlation coefficients: ≥ 0.70) (Rizvi, Peterson, Crow, & Agras, 2000). The EDE adapted for children has shown good interrater reliability (Spearman correlation coefficients: 0.91 to 1.00) and discriminant validity (Cohen's kappa for presence of different eating episode categories of 1.00) in pediatric samples (Glasofer, et al., 2007; Tanofsky-Kraff, et al., 2004; Watkins, Frampton, Lask, & Bryant-Waugh, 2007). Interrater reliability for the presence or absence of loss of control eating among a subset of the current sample (7%) was very good (Cohen's kappa of 0.92). Group consensus was used to determine the episode size when interviewers thought it was ambiguous; this training is reflected in the discriminant validity for the size of different eating episodes in the current study, which was also very good (Cohen's kappa of 1.00).

Standard Pediatric Eating Episode Inventory. The Standard Pediatric Eating Episode Inventory is a semi-structured interview used to assess the behavioral (e.g., eating after consuming a forbidden food, restricting intake prior to eating), physical (e.g., feeling hungry,

full, or sick), and emotional (e.g., negative or positive emotions) aspects surrounding eating episodes (Tanofsky-Kraff, Goossens, et al., 2007). All responses were dichotomous (coded as yes or no). The Standard Pediatric Eating Episode Inventory was administered following the overeating section of the Eating Disorder Examination and the questions were asked regarding the most pathological eating episode for each participant. As previously described (Tanofsky-Kraff, Goossens, et al., 2007), objective binge episodes were considered the most pathological, with subjective binge episodes, objective overeating episodes (unambiguously large amount of food but no loss of control), and normal eating episodes following, respectively. The SPEEI has been shown to differentiate youth with and without loss of control eating (Tanofsky-Kraff, Goossens, et al., 2007).

Depressive Symptoms. The Beck Depression Inventory, Second version (Beck, Steer, & Brown, 1996) is a widely used 21-item self-report measure that assesses depressive symptoms and was administered to a subset of non-intervention participants ≥ 13 years ($n = 187$) and all prevention-seeking girls ($n = 102$). The Beck Depression Inventory has demonstrated excellent psychometric properties (Ambrosini, Metz, Bianchi, Rabinovich, & Undie, 1991; Bennett et al., 1997; Marton, Churchard, Kutcher, & Korenblum, 1991; Spence, Sheffield, & Donovan, 2005). The Children's Depression Inventory, Second version (Kovacs, 1992) was administered to a subset of non-intervention seeking youth ($n = 223$). The Children's Depression Inventory is a 28-item self-report measure that assesses depressive symptoms and is suitable for use in children between 7 to 17 years old. The Children's Depression Inventory has demonstrated good reliability and validity in community and clinical samples (Figueras Masip, Amador-Campos, Gómez-Benito, & del Barrio Gándara, 2010; Sitarenios & Kovacs, 1999; Smucker, Craighead, Craighead, & Green, 1986).

Anxiety Symptoms. The State Trait Anxiety Inventory for Children has a 20-item scale for measuring anxiety proneness as a personality trait (trait anxiety) in youth greater than 8 years (Spielberger, Edwards, Lushene, Montuori, & Platzek, 1973). Children rate anxiety symptoms on a three-point scale: 1 = almost never, 2 = sometimes, and 3 = often. The State Trait Anxiety Inventory for Children has been shown to have good construct validity, internal consistency, and test-retest reliability (Muris, Merckelbach, Ollendick, King, & Bogie, 2002; Perrin & Last, 1992; Spielberger, et al., 1973).

Parent-Reported Behavior Problems. The Child Behavior Checklist (Achenbach & Elderbrock, 1991) was completed by parents to assess their perceptions of their child's internalizing and externalizing symptoms, as well as overall general psychopathology, in the previous 6 months. The Child Behavior Checklist is a 118-item questionnaire designed for use in children between 6-18 years that uses a 3-point scale from "Not true" to "Very true or often true." T-scores were provided to facilitate the interpretation of results. The Child Behavior Checklist is a widely used measure of psychopathology in youth and has shown high internal consistency and test-retest reliability as well as good convergent and discriminant validity (Achenbach & Elderbrock, 1991).

Body Composition. Participants' weight and height were measured in a fasting state using calibrated electronic instruments. BMI was calculated as weight in kilograms divided by the square of height in meters. BMI standard deviation (BMI-z) scores and BMI percentile scores adjusted for age and sex were calculated according to the Centers for Disease Control and Prevention 2000 growth charts (Kuczmarski et al., 2002). Body fat and fat-free mass (kg) was measured either by dual-energy x-ray absorptiometry (DXA) using Hologic QDR-2000 or Hologic QDR 4500A equipment (Hologic, Waltham, MD) or air displacement plethysmography

(Bod Pod; Life Measurement Inc., Concord, CA). As recommended (Nicholson et al., 2001; Robotham et al., 2006), we ensured that measurements of adiposity were equivalent between the two different assessment techniques by adding 2.29 kg to DXA fat mass obtained using the 4500A machine and multiplying girls' Bod Pod fat mass by 1.03.

Assessment of State Negative Affect in the Laboratory. Immediately prior to and following each laboratory test meal, participants completed the well-validated Brunel Mood Scale (Terry, Lane, Lane, & Keohane, 1999), which measures present mood state and generates six subscales pertaining to anger, confusion, depression, fatigue, tension, and vigor. As an overall measure of pre-meal state negative affect, a total score was computed by summing the individual items from the anger, confusion, depression, fatigue, and tension subscales and the reverse-coded items from the vigor subscales. Cronbach's alpha for the Brunel Mood Scale total score in this study was 0.82, indicating very good internal consistency.

Observed Intake During Laboratory Test Meals. A subset of participants from one of the non-intervention study protocols ($n = 223$) was asked to consume their lunch *ad libitum* from a buffet test meal on two separate days. In random order, children and adolescents participated in a "normal meal" (at which they were told to "eat as much as you would at a normal meal") and a "disinhibited" meal (at which they were instructed to "let yourself go and eat as much as you want"). Other than the instruction, all aspects of the conditions were identical. On the day of each test meal, youth were provided with a standard 280 kcal breakfast (7% protein, 19% fat, 74% carbohydrate). Participants remained at the NIH Clinical Center for the next six hours, during which they were observed to ensure that they consumed no calorie-containing foods or beverages and only participated in sedentary activities. Each participant was then presented with a multiple-item, 9,835-kcal food array varied in macronutrient composition (51% carbohydrate,

12% protein, 37% fat, across all foods) and containing a wide assortment of foods (Mirch et al., 2006). Standard amount of foods were presented for breakfast and lunch due to the complexities associated with predicting the amount an individual would eat based on body composition, sex, race, pubertal status, and other environmental factors (e.g., food accessibility). All food items presented were weighed to the nearest 0.1g before and after the test session. Energy content and macronutrient composition were calculated using data from the USDA National Nutrient Database for Standard Reference (USDA, Agricultural Research Service, Beltsville, MD) and food manufacturer information, when available.

Analytic Plan

Data were screened for normality. Outliers were adjusted to fall 1.5 times the interquartile range below or above the 25th or 75th percentile (Behrens, 1997). This strategy was used because it minimizes outliers' influence on the characteristics of the distribution, minimally changes the distribution overall, and avoids potential bias associated with eliminating outliers altogether (Behrens, 1997). After adjusting the outliers, it was confirmed that skew and kurtosis were satisfactory on all variables.

Latent profile analysis (LPA) was used to determine whether children and adolescents can be grouped into subtypes based on the patterns of disinhibited eating behaviors. LPA is an empirically-driven approach that uses categorical and continuous indicators in cross-sectional samples to identify latent classes, or subgroups, of individuals (Crosby et al., 2011). These subgroups are considered latent because membership is not directly observed, but rather it is inferred by examining the patterns of interrelationships among indicator variables (Crosby, et al., 2011). LPA identifies subgroups by maximizing homogeneity within each class and maximizing

heterogeneity between classes. LPA is similar to cluster analysis in that they are both person-centered approaches. However, cluster analysis does not objectively determine the appropriate number of clusters when there is significant overlap among symptoms, which is likely in the current study, and is based on qualitative rather than quantitative distinctions among subgroups of individuals (Crosby, et al., 2011).

The LPA was performed with Latent Gold version 4.5 (Vermunt & Magidson, 2005). Participants were included in the LPA if they completed at least three out of five possible measures of disinhibited eating, which included parental report of eating in the absence of hunger, child report of emotional eating, and three outcomes related to loss of control eating from a semi-structured interview. Five indicators—measures that are thought to operationalize underlying constructs (Crow et al., 2011)—were selected to represent the range of disinhibited eating behaviors, including the: 1) presence of loss of control eating in the previous month; 2) presence of OBEs in the previous month; 3) presence of SBEs in the previous month; 4) Emotional Eating Scale for Children and Adolescents total score; and 5) Parent Report – Eating in the Absence of Hunger total score. The most parsimonious number of latent classes was determined by three fit indices: 1) the Bayesian information criterion (BIC; Schwarz, 1978); 2) the consistent Akaike information criterion (cAIC; Bozdogman, 1987); and 3) the sample size adjusted BIC (aBIC; Sclove, 1987). The lowest value of these fit indices is indicative of the best fitting model. Bivariate residuals, which are indices of the remaining correlations between indicators of disinhibited eating within latent classes, were examined to ensure that none had a value greater than 4.0. After identifying the best fitting model and examining bivariate residuals, participants were assigned to a latent class on the basis of posterior probabilities.

Analyses of covariance (ANCOVA) were used to validate the subtypes identified in the LPA with regard to body composition, eating disorder psychopathology, and general psychopathology. The independent variable was the latent class group assignment. Dependent variables included BMI-z scores, percent fat mass, the Eating Disorder Examination global score and subscales (restraint, and eating-, shape- and, weight- concern), depressive symptoms, trait anxiety scores, and Child Behavior Checklist internalizing, externalizing and total scores. Covariates for all models included age (years), race (coded as non-Hispanic Caucasian or Other), sex (coded as male or female), and prevention-seeking status (coded as presence or absence). Bonferroni-Hochberg post hoc tests were used to examine where the differences were when omnibus tests were significant; the Bonferroni-Hochberg post hoc test was selected to control the family-wise error rate when conducting multiple comparisons. Pearson chi-square tests were used to validate the subtypes based on the overlap of disinhibited eating behaviors with regard to SPEEI variables. Since the prevention girls might disproportionately influence findings because of the restricted BMI percentile range, reports of loss of control eating, and any additional distress associated with seeking intervention, all body composition and psychological analyses were repeated with only the non-intervention participants.

For the subset of non-treatment-seeking youth who participated in the laboratory test meals, linear mixed models with repeated measures were used to examine the relationship between disinhibited eating behavior subtypes and observed energy intake. The repeated measure was meal instruction (normal versus disinhibited). The dependent variables were total energy intake (kcal; logarithm transformed), percent macronutrient content consumed (carbohydrate, fat, and protein; arcsine transformed), and pre- and post-meal ratings of state negative affect. The independent variable was the latent class group assignment. In a second analysis, meal

instruction (normal versus disinhibited) was also included as an independent variable to test the two-way interaction of meal instruction and latent class group assignment. Covariates included age (years), race (coded as non-Hispanic Caucasian or other), sex (coded as male or female), height (cm), lean mass (kg), and percent fat mass (%). Total energy consumed (kcal; logarithm transformed) was included as a covariate in the models examining percent macronutrient content intake (arcsine transformed) and post-meal state negative affect. Pre-meal negative affect was included as a covariate in the model examining post-meal negative affect.

For all tests examining the validity of the subtypes generated by the LPA, SPSS version 19.0 was used. Associations were considered significant when p values were ≤ 0.05 . All tests were two-tailed.

Chapter 4: Results

Data from 512 children and adolescents (8 to 18 years, $M = 14.11$ years, $SD = 2.41$; 61% female) were included in the LPA. Four hundred-ten participants (80.1%) were enrolled in the non-intervention studies and 102 girls (19.9%) were in the prevention protocol. The racial/ethnic breakdown of the sample was 55.3% non-Hispanic White, 30.3% non-Hispanic Black or African American, 4.5% Asian Origin, 1.8% Hispanic/Latino, 2.5% Multiple Races, and 5.6% Other/Unknown. Youth represented a wide range of weight strata (BMI-z score: Range = -2.19 to 3.20, $M = 0.94$, $SD = 1.05$).

Latent Profile Analysis: Characterization of Disinhibited Eating Subtypes

The extent of missing data for each indicator variable in the LPA models were as follows: 0.4% for LOC presence ($n = 2$), 1.8% ($n = 9$) for OBE presence, 0.4% ($n = 2$) for SBE presence, 17.0% ($n = 87$) for emotional eating, and 13.7% ($n = 70$) for eating in the absence of hunger. LPA models were evaluated with the number of clusters ranging from one to eight. All bivariate residuals were less than 4.0, indicating that the conditional independence assumption was met. The BIC, aBIC, and cAIC were lowest for a four-cluster model with 31 parameters (**Table 1**). The relative frequency of loss of control eating (OBE and SBE), objective binge episodes (OBEs), and subjective binge episodes (SBEs) and relative severity of parent-reported eating in the absence of hunger and self-reported emotional eating for each of the four clusters is depicted in **Figure 2**.

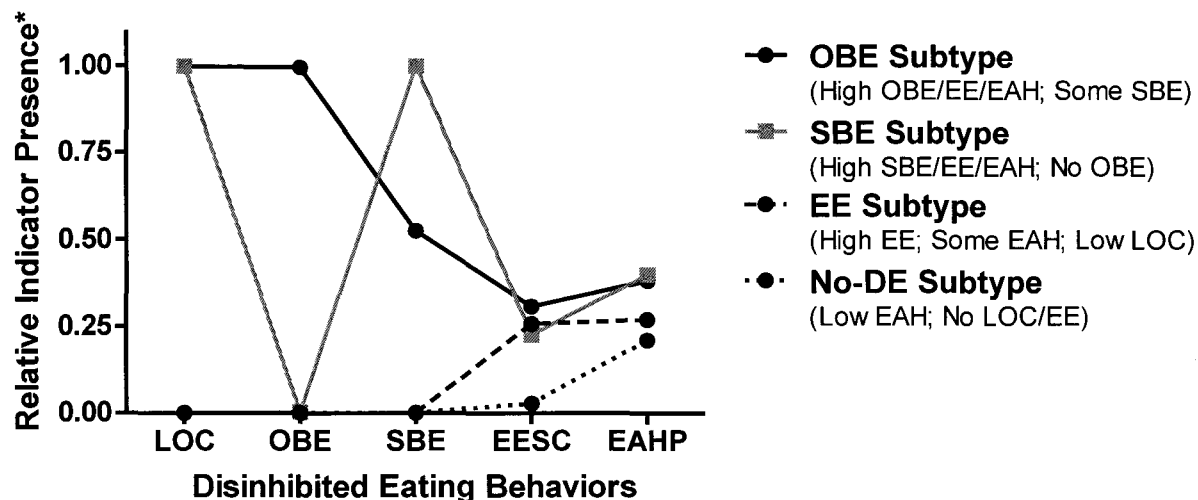
Cluster 1 comprised 25.9% ($n = 133$) of the sample and resembles an asymptomatic cluster with little to no disinhibited eating behaviors. Among youth in Cluster 1, none reported

Table 1. Fit Indices for the Latent Profile Analysis Determining the Typology of Pediatric Disinhibited Eating Behaviors.

Clusters	Parameters	BIC	aBIC	cAIC	LL
1	7	6223.49	6201.27	6230.49	-3089.91
2	15	5552.66	5505.02	5567.66	-2729.53
3	23	5491.50	5418.44	5514.50	-2673.98
4	31	5442.90	5344.45	5473.90	-2624.73
5	39	5454.24	5330.38	5493.24	-2605.44
6	47	5453.76	5304.48	5500.76	-2580.23
7	55	5465.53	5290.85	5520.53	-2561.16
8	63	5488.97	5288.86	5551.97	-2547.91

Note: BIC = Bayesian Information Criterion; cAIC = Consistent Akaike Information Criterion; aBIC = sample size adjusted BIC; LL = Log-likelihood; Lower BIC, aBIC, and cAIC values indicate better model fit.

Figure 2. Relative Frequency of Latent Profile Analysis Indicators Among Disinhibited Eating Subtypes.



Note: The figure depicts the relative frequency of the five indicator variables for the four disinhibited eating subtypes that emerged from the latent profile analysis. LOC = Loss of control eating; OBE = Objective binge episode; SBE = Subjective binge episode; No-DE = No disinhibited eating; EESC = Emotional Eating Scale for Children and Adolescents; EAHP = Eating in the Absence of Hunger Scale – Parent Version.

*LOC, OBE, and SBE reflect the percentage of youth in each subtype reporting the presence of each indicator. EESC and EAHP scores represent the standardized means for each subtype.

any episodes of loss of control eating and nearly non-existent levels of emotional eating were reported; however, there were low levels of eating in absence of hunger reported. Cluster 2 comprised 18.3% (n = 93) of the sample and resembled a group with moderate levels of emotional eating and eating in the absence of hunger. None of the youth in Cluster 2 reported the presence of loss of control eating (OBE or SBE). Cluster 3 comprised 40.4% (n = 207) of the sample and is characterized by the presence of loss of control eating, specifically the presence of SBEs, moderate levels of emotional eating, and the highest severity of eating in the absence of hunger. All youth in Cluster 3 reported SBEs, while none reported OBEs. Cluster 4 comprised 15.4% (n = 79) of the sample and resembles a group characterized by the presence of loss of control eating and a high severity with regard to eating in the absence of hunger and emotional eating. All youth in Cluster 4 reported the presence of OBEs and 52% also reported the presence of SBEs. For ease of discussion, the clusters will be referred based on their most distinguishing characteristic: 1) Cluster 1 as the “No Disinhibited Eating (No-DE)” subtype; 2) Cluster 2 as the “Emotional Eating (EE)” subtype; 3) Cluster 3 as the “Subjective Binge Episode (SBE)” subtype; and 4) Cluster 4 as the “Objective Binge Episode (OBE)” subtype.

Disinhibited Eating Subtypes and Clinical Characteristics

Demographics, body composition, and psychological variables based upon disinhibited eating subtypes with regard to are summarized in **Table 2**. There were no differences among the subtypes with regard to age or racial/ethnic background. Due to the prevention study being comprised of only girls with loss of control eating patterns, those in the OBE and SBE subtypes were more likely to be female as compared to those in the EE and No-DE Subtypes ($p = .01$). Youth in the OBE and SBE subtypes had significantly greater BMI-z scores and higher overall

Table 2. Disinhibited Eating Subtype Differences with Regard to Demographic Variables, Body Composition, and Eating Disorder and General Psychopathology.

Variable	OBE Subtype (n = 79)		SBE Subtype (n = 207)		EE Subtype (n = 93)		No-DE Subtype (n = 133)		Test statistic	P value	Effect Size ^a
	M	SD	M	SD	M	SD	M	SD			
Age (years)	14.11	2.53	14.12	2.19	14.33	2.46	16.63	2.46	F(3, 486) = 1.80	.15	.01
BMI-z	1.24 _a	0.93	1.37 _a	0.76	0.63 _b	1.09	0.75 _b	1.12	F(3, 494) = 17.33	<.001	.10
Total fat mass (%)	32.59 _a	11.92	34.82 _a	9.69	24.11 _b	12.08	24.25 _b	11.59	F(3, 473) = 7.65	<.001	.05
EDE Global Score	1.12 _a	0.84	1.14 _a	0.76	0.36 _b	0.45	0.35 _b	0.46	F(3, 476) = 18.94	<.001	.11
Depressive Symptoms ^b	9.42 _a	6.14	8.49 _{ab}	5.98	4.81 _b	4.47	3.17 _b	3.44	F(3, 250) = 5.11	.002	.06
State Anxiety	35.81 _a	6.20	33.19 _b	6.95	29.16 _c	6.23	28.18 _c	6.52	F(3, 291) = 10.51	<.001	.10
CBCL Total Problems	49.74 _a	11.27	50.51 _a	12.17	46.27 _a	10.98	41.97 _b	11.35	F(3, 458) = 3.62	.01	.02
	n	%	n	%	n	%	N	%			
Female	60	75.95 _a	114	85.71 _a	101	49.03 _b	37	39.36 _b	χ^2 (3, n = 512) = 75.37	<.001	.38
Non-White Ethnicity	37	46.84	62	46.61	85	41.26	45	47.87	χ^2 (3, n = 512) = 1.71	.64	.06

Note: Subscripts that differ represent differences between the subtypes using a Bonferroni-Hochberg post-hoc test. OBE = Objective binge episode; SBE = subjective binge episode; EE = Emotional eating; No-DE = No disinhibited eating; EDE = Eating Disorder Examination; CBCL = Child Behavior Checklist (parent-reported psychopathology).

^a Effect sizes for analyses of variance are partial η^2 ; effect sizes for chi-square tests are ϕ^2 .

percent fat mass than youth in the EE and No-DE subtypes ($ps < .001$). Findings with regard to sex ($p = .004$), BMIz ($p = .002$), and percent fat mass ($p = .02$) based upon subtype remained the same when examining only the non-intervention participants.

Youth in the OBE and SBE subtypes reported higher global eating disorder psychopathology than those in the EE and No-DE subtypes ($p < .001$). This pattern of results was the same for restraint, $F(3, 478) = 7.41, p < .001, \eta^2 = .05$, shape concern, $F(3, 476) = 18.62, p < .001, \eta^2 = .11$, and weight concern, $F(3, 476) = 8.43, p < .001, \eta^2 = .05$. Youth in the OBE subtype reported the greatest eating concern ($M = 0.63, SD = 0.73$) as compared to all other subtypes, $F(3, 476) = 20.55, p < .001, \eta^2 = .12$. Those in the SBE subtype ($M = 0.44, SD = 0.53$) reported higher eating concern than those in the EE subtype ($M = 0.09, SD = 0.23$) and No-DE subtype ($M = 0.07, SD = 0.22$). When the prevention participants were removed, all findings persisted.

Differences among the disinhibited eating subtypes also emerged with regard to measures of general psychopathology. Youth in the OBE subtype reported greater depressive symptoms, as assessed by the Beck Depression Inventory ($n = 289$), than those in the EE and No-DE subtypes ($ps < .01$). The SBE subtype did not significantly differ from any subtypes with regard to Beck Depression Inventory scores. Among youth who completed the Children's Depression Inventory ($n = 223$), the OBE subtype ($M = 8.55, SD = 8.12$) reported higher depressive symptoms than all other subtypes; the SBE subtype ($M = 6.69, SD = 5.45$) reported significantly greater depressive symptoms than the EE ($M = 4.71, SD = 4.16$) and No-DE subtypes ($M = 3.66, SD = 3.80$), which did not differ from each other, $F(3, 215) = 6.92, p < .001, \eta^2 = .09$. Trait anxiety was highest among those in the OBE subtype; youth in the SBE subtype reported higher trait anxiety than both the EE and No-DE subtypes ($ps < .001$). As compared to those in the No-DE subtype, youth

in the OBE, SBE, and EE subtypes had higher overall general psychopathology as reported by their parents ($p = .01$). There were no significant subtype differences in relation to parent-reported internalizing, $F(3, 458) = 2.50, p = .06, \eta^2 = .02$, and externalizing problem behaviors, $F(3, 458) = 1.58, p = .19, \eta^2 = .01$. Once again, when we only studied the non-intervention participants, results remained the same.

Disinhibited Eating Subtypes and the Context of an Aberrant Eating Episode

The comparison of disinhibited eating subtypes with regard to affect and behaviors that occur before, during, and after a reported aberrant eating episode is summarized in **Table 3**. Compared to youths in the EE and No-DE subtypes, greater percentages of participants in the OBE and SBE subtypes reported a trigger before eating, $\chi^2(3, n = 382) = 19.11, p < .001, \phi = .22$, eating at a faster rate than normal during the episode, $\chi^2(3, n = 384) = 17.23, p = .001, \phi = .22$, feeling secretive during the episode, $\chi^2(3, n = 289) = 27.22, p < .001, \phi = .31$, dissociation during the episode, $\chi^2(3, n = 380) = 53.72, p < .001, \phi = .38$, and feeling guilt or shame after the episode, $\chi^2(3, n = 384) = 13.13, p = .002, \phi = .20$. Youths in the OBE and SBE subtypes also had greater percentages of negative affect reports before the episode began as compared to those in EE and No-DE subtypes, $\chi^2(3, n = 382) = 12.58, p = .01, \phi = .18$. Additionally, those in the EE subtype reported higher percentages of experiencing negative affect before the episode as compared to the No-DE subtype. This same pattern of results emerged for eating in the absence of hunger during the episode, $\chi^2(3, n = 382) = 39.48, p < .001, \phi = .32$, in which OBE and SBE subtypes had the highest percentages followed by the EE subtype and No-DE subtype, respectively. Compared to all other subtypes, youths in the SBE subtype were more likely to report restricting before the eating episode began, $\chi^2(3, n = 385) = 9.05, p = .03, \phi = .15$. Youths

Table 3. Disinhibited Eating Subtype Differences with Regard to Affect and Behavior Before, During, and After a Reported Eating Episode.

Variable	OBE Subtype		SBE Subtype		EE Subtype		No-DE Subtype		Test statistic	P value	Effect Size (φ)
	n	%	n	%	n	%	N	%			
Before Eating Episode											
Hiding Food	5	7.24	5	4.38	3	2.11	0	0.00	$\chi^2 (3, n = 380) = 6.06$.11	.13
Restricting	10	14.49 _a	32	27.82 _b	21	14.48 _a	9	16.07 _a	$\chi^2 (3, n = 385) = 9.05$.03	.15
Negative Affect	14	20.90 _a	25	21.74 _a	17	11.72 _b	2	3.64 _c	$\chi^2 (3, n = 382) = 12.58$.01	.18
Trigger	16	23.18 _a	24	20.87 _a	11	7.69 _b	2	3.64 _b	$\chi^2 (3, n = 382) = 19.11$	<.001	.22
During Eating Episode											
Fast Eating	38	55.88 _a	48	41.74 _a	43	29.66 _b	15	26.79 _b	$\chi^2 (3, n = 384) = 17.23$.001	.21
Ate More Than Others	21	31.34	35	29.66	41	29.08	17	29.82	$\chi^2 (3, n = 383) = 0.11$.99	.02
Eating in the Absence of Hunger	50	73.53 _a	71	62.28 _a	67	46.21 _b	12	21.82 _c	$\chi^2 (3, n = 382) = 39.48$	<.001	.32
Feeling Secretive	5	11.63 _a	8	16.67 _a	0	0.00 _b	1	1.82 _b	$\chi^2 (3, n = 289) = 27.22$	<.001	.31
Dissociation	24	35.82 _a	37	32.17 _a	8	5.59 _b	1	1.82 _b	$\chi^2 (3, n = 380) = 53.72$	<.001	.38
After Eating Episode											
Negative Affect	40	61.54 _a	88	78.57 _b	26	18.18 _c	10	18.18 _c	$\chi^2 (3, n = 375) = 116.15$	<.001	.56
Guilt or Shame	22	32.84 _a	32	28.83 _a	21	14.48 _b	9	16.67 _b	$\chi^2 (3, n = 384) = 13.13$.004	.19
Feeling Guilty	18	26.00	24	20.87	11	7.69	11	19.64	$\chi^2 (3, n = 377) = 14.68$.002	.20

in the SBE subtype were the most likely to report negative affect after an episode, $\chi^2 (3, n = 375) = 116.15, p < .001, \phi = .56$. Those in the OBE subtype were more likely to report negative affect after an episode than those in the EE and No-DE subtypes, which did not differ from each other. As compared to the OBE, SBE, and No-DE subtypes, youths in the EE subtype were less likely to feel sick after an episode, $\chi^2 (3, n = 377) = 14.68, p < .001, \phi = .20$. There were no subtype differences with regard to hiding food before the episode, $\chi^2 (3, n = 380) = 6.06, p = .11, \phi = .13$, or the amount consumed as compared to others, $\chi^2 (3, n = 383) = 0.11, p = .99, \phi = .02$. Results remained the same when examining only non-intervention participants.

Disinhibited Eating Subtypes and Eating Behavior in the Laboratory

A subset ($n = 223$) from the non-intervention sample participated in the two test meals. Differences in disinhibited eating subtypes and mood and eating behavior in the laboratory are depicted in **Table 4**. After controlling for sex, age, race, height (cm), lean mass (kg), and percent fat mass (kg), there was a main effect of disinhibited eating subtype on total energy intake, $F(3, 244.64) = 3.74, p = .01$. Specifically, the SBE subtype consumed less overall energy than the OBE and EE subtypes, and the No-DE subtype did not differ from the SBE, OBE, or EE subtypes. Regardless of meal type, youth in the OBE and SBE subtypes consumed a smaller percentage of energy from protein, $F(3, 246.47) = 3.62, p = .01$, and a larger percentage of energy from carbohydrate, $F(3, 247.02) = 2.93, p = .03$, than the EE and No-DE subtypes. There was no main effect of disinhibited eating subtype on percentage of energy from fat consumed, $F(3, 246.42) = 1.66, p = .18$. Youth in the OBE, SBE, and EE subtypes reported greater state negative affect before the test meals than the No-DE subtype, after accounting for relevant covariates, $F(3, 420.96) = 11.62, p < .001$. There was no main effect of subtype in post-meal

state negative affect, $F(3, 239.78) = 1.73, p = .16$. There was no interaction of meal type with disinhibited eating subtype for any of the models ($ps = .13$ to $.36$).

Table 4. Disinhibited Eating Subtype Differences with Regard to State Negative Affect and Eating Behavior in the Laboratory.

Variable	OBE Subtype (n = 41)		SBE Subtype (n = 35)		EE Subtype (n = 113)		No-DE Subtype (n = 44)		Test statistic	P value
	M	SE	M	SE	M	SE	M	SE		
Pre-meal negative affect	6.76 _{ab}	0.56	7.03 _a	0.49	5.42 _b	0.33	3.16 _c	0.49	$F(3,420.96)=11.6$	<.00
Post-meal negative affect	3.62	0.27	3.63	0.25	3.15	0.16	2.96	0.24	$F(3,239.78)=1.7$.16
Total intake	1470.82 _a	66.36	1246.60 _b	55.11	1495.22 _a	42.77	1369.68 _a	63.49	$F(3,244.64)=3.7$.01
% Protein	13.70 _a	0.63	13.95 _a	0.62	15.15 _b	0.33	15.32 _b	0.49	$F(3,246.47)=3.6$.01
% Fat	36.60	1.10	37.43	1.09	37.96	0.58	38.58	0.87	$F(3,244.87)=1.4$.18
% Carbohydrate	51.20 _a	1.44	49.95 _a	1.43	48.06 _b	0.76	47.18 _b	1.13	$F(3,245.62)=2.9$.03

Note: N = 223. Means and standard errors are presented as a representation from both test meals. Subscripts that differ represent differences between the subtypes using a Bonferroni-Hochberg post-hoc test. OBE = Objective binge episode; SBE = subjective binge episode; EE = Emotional eating; No-DE = No disinhibited eating.

Chapter 5: Discussion

This study made use of latent profile analysis (LPA) to identify four subtypes based on the overlap of eating in the absence of hunger, emotional eating, and loss of control eating. Results indicated substantial overlap among these disinhibited eating behaviors. However, the subtypes were characterized based on distinguishing features based on “objective binge episode (OBE),” “subjective binge episode (SBE),” “emotional eating (EE),” and “no disinhibited eating (No-DE).” Findings suggest that these subtypes can be differentiated on measures of adiposity, psychological functioning, and objective measures of eating behavior in the laboratory, such that the loss of control subtypes were the most pathological in terms of reported and objective measures. Our findings highlight the importance of considering the entire range of disinhibited eating behaviors in youth and point towards potential targeted intervention strategies. Moreover, this study underscores the tremendous utility of LPA in examining subthreshold symptoms that hold potential importance for preventative and early intervention efforts.

Overall, the findings support the proposed model that disinhibited eating behaviors are likely organized in a pyramidal fashion (Shomaker, Tanofsky-Kraff, & Yanovski, 2010). This model suggests that disinhibited eating behaviors are ordered from bottom to top based upon prevalence, pathology, and overlap (see **Figure 1**). Eating in the absence of hunger—at the bottom of the pyramid—is hypothesized to be the most prevalent and least pathological disinhibited eating behavior reported among children and adolescents. Emotional eating is thought to be slightly less common and somewhat more pathological, while loss of control eating (both objective and subjective binge episodes) —at the top of the pyramid—is proposed to occur among the smallest subset of youth and to be associated with the most severe psychological and

weight-related associations and outcomes. Also important to the pyramid model, is that behaviors at the top (loss of control eating) often include behaviors in lower parts of the pyramid (emotional eating and eating in the absence of hunger). In contrast, behaviors at the bottom (eating in the absence of hunger) do not necessarily involve behaviors located above them (e.g., emotional eating and loss of control eating).

In summary, findings from the current study provide support for the predictions of the pyramid model with regard to the hypothesized overlap and pathology relationships. With regard to the hypothesized overlap of disinhibited eating behaviors, subsets of youth were identified who reported: 1) only marginal levels of eating in the absence of hunger; 2) both emotional eating and eating in the absence of hunger but not loss of control eating; and 3) all three disinhibited eating behaviors. Our findings also support the pyramid model's hypothesized pathology associated with different patterns of disinhibited eating behavior overlap. For example, the presence of all three disinhibited eating behaviors in youth was consistently associated with greater adiposity, aberrant eating behavior in the laboratory, and higher disordered eating attitudes and general psychopathology as compared to those with two disinhibited eating behaviors. However, our data also suggest that there may be differences among individuals reporting OBEs versus SBEs—two types of loss of control eating, which was not hypothesized by the pyramid model. Further, our pyramid model was not supported with regard to the prevalence of behaviors.

Detailed Discussion of Findings

The No-DE subtype was characterized by youth who were reported to eat in the absence of hunger occasionally and had no emotional eating or loss of control eating. Notably, despite a

wealth of research on eating in the absence of hunger (Cutting, et al., 1999; Fisher & Birch, 2002; Fisher, et al., 2007; Hill, et al., 2008; Moens & Braet, 2007; Shomaker, Tanofsky-Kraff, Zocca, et al., 2010; Zocca, et al., 2011), a distinct subtype distinguished primarily by eating in the absence of hunger did not emerge. Indeed, it is possible that the contemporary obesogenic environment creates conditions in which the vast majority of children and adolescents—at least occasionally—rely on external food cues rather than internal hunger and satiety signals (Wardle, 2005). Therefore, eating in the absence of hunger—when considering other disinhibited eating behaviors—may not be particularly clinically meaningful. In support of this notion, a prospective link between eating in the absence of hunger and obesity has yet to be demonstrated. However, it is possible that eating in the absence of hunger in vulnerable youth may elevate risk for the development of more pathological disinhibited eating behaviors, chronic overeating, and obesity.

Children and adolescents comprising the EE subtype reported moderately high levels of emotional eating and sometimes eating in the absence of hunger, but no loss of control eating. Moreover, the EE subtype was more pathological than the No-DE subtype but less pathological than the two subtypes characterized by loss of control eating. Specifically, youth with the EE subtype had higher parent-reported behavior problems and had greater state negative affect prior to the test meals as compared to those with the No-DE subtype. These findings indicate that the presence of emotional eating in pediatric samples may be an indicator of some emerging mood and behavior problems. Fortunately, our data also suggest that these youth were not yet engaging in disordered eating behavior or experiencing disordered eating attitudes. Given that emotional eating has been shown to predict binge eating onset in adolescents (Stice, et al., 2002), emotional eating may represent an important point of early prevention during childhood.

Two subtypes emerged from the LPA that were differentiated by the presence of loss of control eating. The OBE subtype consisted of youth who all reported consuming unambiguously large amounts of food while experiencing a sense of loss of control. In contrast, the SBE subtype had children and adolescents who reported the presence of only ambiguously large binge episodes. Both subtypes were also characterized by higher levels of emotional eating and eating in the absence of hunger than the EE and No-DE subtypes. These findings are comparable to previous studies indicating an overlap between these disinhibited eating behaviors (Goossens, et al., 2007; Moens & Braet, 2007; Shomaker, Tanofsky-Kraff, Elliott, et al., 2010; Tanofsky-Kraff, et al., 2008; Tanofsky-Kraff, Theim, et al., 2007; Zocca, et al., 2011).

When considering patterns of overlap among all three types of disinhibited eating behaviors, results suggest that loss of control eating may be the most potent behavioral indicator of adverse eating- and weight-related problems in children and adolescents. In comparison to the EE and No-DE subtypes, the loss of control eating subtypes had greater BMI_z scores and percent fat mass, reported higher eating disorder psychopathology and general psychological distress, and consistent with prior research (Hilbert, et al., 2010; Tanofsky-Kraff, McDuffie, et al., 2009) consumed test meals that were lower in protein and higher in carbohydrate composition. In line with previous findings (Shomaker, Tanofsky-Kraff, Elliott, et al., 2010), the OBE and SBE subtypes did not often significantly differ from each other on many measures. Despite similarities between the loss of control eating subtypes, there were also marked differences. Youth comprising the OBE subtype reported higher trait anxiety and eating concerns than those in the SBE subtype. The OBE subtype was also associated with higher depressive symptoms than the EE and No-DE subtypes, whereas the SBE subtype did not differ from any of the subtypes on this outcome.

The differences between the OBE and SBE subtypes point towards important clinical implications. These data suggest that the presence of objective binge episodes alone may be associated with elevated psychopathology that warrants more focus during treatment compared to other profiles of disinhibited behaviors. The SBE subtype consumed approximately 220 kcal less overall energy in the laboratory as compared to other disinhibited eating subtypes. If these results generalize to the natural environment, the SBE subtype may be less prone to the excessive weight gain associated with loss of control eating. However, results from retrospective reports of behaviors prior to an LOC episode suggest that it is also possible that youth with the SBE subtype overeat following periods of restriction, and may explain why loss of control episodes, regardless of episode size, promote excess weight gain (Tanofsky-Kraff, Yanovski, et al., 2009). In terms of clinical implications, it is possible that cognitive behavior therapy, which is based on the restraint theory of binge eating (Polivy & Herman, 1985), may be more appropriate for youth with a disinhibited eating profile similar to the SBE subtype. Longitudinal studies are needed to examine the long-term eating and weight outcomes of these disinhibited subtypes distinguished by loss of control eating.

The pyramid model of disinhibited eating hypothesized that disinhibited eating behaviors at the top of the pyramid would be less prevalent in the population than behaviors towards the bottom of the pyramid. The LPA subtype breakdown by the entire sample ($N = 512$) for the current study was: 25.9% for the No-DE subtype, 18.4% for the EE subtype, 40.3% for the SBE subtype, and 15.4% for the OBE subtype. Although these findings do not fully support the prevalence predictions made by the pyramid model, which represented approximately 40% of the sample. As compared to the general population, it is likely that the presence of LOC eating was overrepresented in the current study because intervention-seeking girls with LOC eating were

included. This may account for the disproportionate number of youth in the SBE subtype. Among only non-intervention participants ($n = 410$), the LPA subtype breakdown was: 22.7% for the No-DE subtype, 50.0% for the EE subtype, 14.4% for the SBE subtype, and 12.9% for the OBE subtype. While the relative frequency of disinhibited eating subtypes in the non-intervention sample is closer to the hypotheses of the pyramid model, there is still a small number of participants in the No-DE subtype than expected. It is possible that the recruitment methods for the study, which targeted youth who were specifically interested in studies on eating behaviors, may have biased the sample towards participants with some aberrant eating. Large-scale, nationally representative studies are needed to determine the prevalence of disinhibited eating subtypes in the general population.

Future Directions

Overall, findings from the current study provide compelling support for distinct disinhibited eating subtypes. Therefore, it seems prudent for clinicians to assess for the presence of all disinhibited eating behaviors in order to obtain a more comprehensive clinical picture of behavioral risk factors for eating and weight problems. Future work may benefit from studying these behaviors from a developmental perspective. That is, it is possible that some disinhibited eating behaviors emerge earlier than others. Studies have shown that eating in the absence of hunger can be observed in children as young as four years old (Fisher & Birch, 2002; Fisher, et al., 2007), whereas emotional eating is rarely reported in early childhood (Carper, et al., 2000; Wardle, et al., 2001) and starts to be seen with greater frequency during middle childhood (Shapiro, et al., 2007; Tanofsky-Kraff, Theim, et al., 2007; van Strien & Oosterveld, 2008). Retrospective reports in adults with binge eating disorder (Abbott et al., 1998; Grilo & Masheb,

2000; Spurrell, Wilfley, Tanofsky-Kraff, & Brownell, 1997) and overweight children (Tanofsky-Kraff, et al., 2005) suggest that loss of control eating may begin between middle childhood and early adolescence. It is conceivable that eating in the absence of hunger may increase risk for the development of emotional eating and subsequently binge eating in youth. Prospective studies that assess all of these disinhibited eating behaviors are needed to clarify the temporal relationship between the development of these behaviors.

It is also important for future research to consider biological mechanisms that may help to explain some of the common mechanisms by which disinhibited eating behaviors are developed and maintained in a pyramidal fashion (**Figure 1**). Preliminary genetic findings point toward shared biological underpinnings that may account for the proposed overlap among disinhibited eating behaviors. The presence of at least one polymorphism in the rs9939609 SNP of the *FTO* gene—an allelic variant known to be tied to obesity and satiety centers in the brain (Dina et al., 2007; Frayling et al., 2007)—are related to greater eating in the absence of hunger and parental reports of satiety responsiveness in young children (Wardle et al., 2008; Wardle, Llewellyn, Sanderson, & Plomin, 2009) as well as the presence of loss of control eating in children and adolescents (Tanofsky-Kraff, Han, et al., 2009). These findings persisted even after controlling for body weight, suggesting that the *FTO* risk allele may exert its effects on the emergence of eating in the absence of hunger and loss of control eating via altering children’s trait-like tendency to become aware of, recognize, or respond to physiological sensations of hunger and fullness. The *TaqA1* allele of the *DRD2* gene—associated with impaired dopaminergic signaling in neural reward pathways—has been associated with the presence of binge eating disorder in adults (Davis et al., 2009) and also reports of emotional eating in adolescents (van Strien, Snoek, van der Zwaluw, & Engels, 2010). Therefore, such disinhibited eating behaviors might reflect a

common susceptibility to reliance on emotional cues to trigger intake or to a heightened sensitivity for food rewards. In spite of these encouraging initial data, much still remains to be determined in regard to the specific mechanisms by which genetic risk alleles influence the development, overlap, and clinical significance of disinhibited eating behaviors. Future work would likely benefit from studies of genes related to the serotonergic and opioidergic systems in the brain, which are thought to be related to the pleasurable experience or “liking” of palatable foods. Longitudinal neuroimaging studies of youth across the disinhibited eating subtypes would also shed light on potential neurobiological mechanisms associated with the onset and maintenance of these behaviors over time.

To inform the development of early preventive interventions, research is needed to identify psychosocial and environmental risk and protective factors common to all disinhibited eating behaviors as well as those unique to the development of more pathological forms of disinhibited eating. Overall, data support a role for parental feeding practices in the development of children’s disinhibited eating behaviors. Infants who were exclusively breastfed during their first six months of life were rated by parents as being less likely to fully empty a bottle or “eat in the absence of hunger” than those who were not exclusively breastfed (Li, Fein, & Grummer-Strawn, 2010), which may be due to higher levels of appetitive hormones (e.g., leptin) present in breastmilk (Palou & Pico, 2009). Additionally, reported parental restriction of child food intake was prospectively linked to increases in observed eating in the absence of hunger in young girls (Birch, et al., 2003; Fisher & Birch, 2002) and cross-sectionally associated with greater reports of emotional eating in children (Carper, et al., 2000; van Strien & Bazelier, 2007; van Strien & Oosterveld, 2008). In one observational study of emotional eating, children ate more snack foods after exposure to a negative (compared to a neutral) mood induction, but only if their parents

reportedly encouraged their children to use food to cope with emotions (Blissett, Haycraft, & Farrow, 2010). Perceived parental monitoring of food intake was associated with a decreased likelihood of emotional eating in children (Farrow, 2012), and reporting of regular family dinners was found to be a protective factor for the onset of OBEs in adolescents (Haines, Gillman, Rifas-Shiman, Field, & Austin, 2010). While there has been limited research focused on factors outside of the parent-child context that influence the development of eating in the absence of hunger or emotional eating, there is some data to suggest that risk factors for LOC eating include dieting or extreme weight control behaviors (Haines & Neumark-Sztainer, 2006; Neumark-Sztainer et al., 2007; Stice, 1998, 2001; Stice, et al., 2002), a history of negative weight-related comments from family and peers (Field et al., 2008; Haines, Neumark-Sztainer, Eisenberg, & Hannan, 2006), perceived sociocultural pressure to be thin from the media and interpersonal sources (parents and peers) (Stice, et al., 2002), and parental and peer modeling of body image dissatisfaction or disordered eating behavior (Stice, et al., 2002). Similar to studies focused on the clinical significance of disinhibited eating behaviors, prior risk and protective factor studies for eating in the absence of hunger, emotional eating, and loss of control eating primarily have been conducted in isolation. Examination of shared and unique factors associated with the development of all three disinhibited eating behaviors is warranted.

Strengths and Limitations

A strength of this study is the application of LPA to investigate the typology of pediatric disinhibited eating behaviors. There are no known studies that have used this empirical classification method to characterize the pattern of subthreshold disordered eating symptoms with a focus on early identification and prevention of risk behaviors. Results also remained the

same in all analyses when investigating the combined sample (non-intervention and prevention studies) and the non-intervention sample only; this increases the generalizability to both treatment-seeking and non-treatment seeking youth. Additional study strengths include the large and diverse sample of children and adolescents spanning a broad weight range and the use of interview assessment of eating disorder psychopathology. While the objective assessment of energy intake at a test meal designed to capture disinhibited eating represents an important strength, there may also be limited generalizability of these findings to the natural environment. Future studies of disinhibited eating behaviors would benefit from using ecological momentary assessment to capture affect and eating patterns. Another limitation is the cross-sectional design that limits our capacity to make causal inferences; longitudinal studies of disinhibited eating behaviors are required. Finally, the nature of the recruitment methods that targeted children interested in eating behaviors studies may limit generalizability to the general population. Likewise, the inclusion of prevention-seeking adolescent girls may have influenced our results. However, findings remained significant after accounting for prevention-seeking status and only analyzing data from the non-intervention participants.

Conclusions

In conclusion, this study suggests that LPA can be used to identify subtypes based on the overlap of eating in the absence of hunger, emotional eating, and loss of control eating in children and adolescents. These findings highlight the heterogeneity of youth at risk for obesity and eating disorders. The presence of loss of control eating may be the most compelling indicator of eating- and weight-related problems in youth, while emotional eating may signal early signs of psychopathology. Within the context of other disinhibited eating behaviors, eating in the

absence of hunger does not appear to be clinically meaningful in children and adolescents. Youth reporting loss of control eating along with other disinhibited eating behaviors may require treatment targeting the elevated eating and weight problems, while prevention interventions may be suitable among those only reporting emotional eating and eating in the absence of hunger. Most importantly, prospective research is required to determine whether disinhibited eating subtypes are predictive of distinct eating and weight outcomes over time.

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